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Chemical Contamination of Water Supplies

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Man-made organic chemicals have been found in drinking water for many years. Their numbers and varieties increase as our analytical capabilities improve. The identified chemicals comprise 10 to 20% of the total organic matter present. These are volatile or low molecular weight compounds which are easily identified. Many of them are carcinogenic or mutagenic. Chlorinated compounds have been found in untreated well water at levels up to 21,300 μg/L and are generally present at higher levels in chlorine-treated water than in untreated water.

Aggregate risk studies for cancer are summarized. The most common sites are: bladder, stomach, colon, and rectum. Such studies cannot be linked to individual cases. However, they are useful for identifying exposed populations for epidemiologic studies. Five case-control studies were reviewed, and significant associations with water quality were found for: bladder cancer in two studies, colon cancer in three and rectal cancer in four.

A large study by the National Cancer Institute found that there had been a change in the source of raw water for 50% of the persons in one area between the years 1955 and 1975. Such flaws in the data may preclude finding a causal relation between cancer and contaminants in drinking water. Large case-control and cohort studies are needed because of the low frequency of the marker diseases, bladder and rectal cancer. Cohort studies may be precluded by variations in the kinds of water contaminants. Definitive questions about these issues are posed for cooperative effort and resolution by water chemists, engineers, and epidemiologists.

Organic chemicals of anthropogenic sources are widely found in drinking water supplies. The problem is not a new one. In 1956, Middleton and Rosen (*1*) examined raw and finished water from five midwestern U.S. cities and reported the presence of benzene compounds, insecticides, kerosene, phenols, polycyclic hydrocarbon compounds, and synthetic detergents. With increasing sophistication of methods for chemical analysis, it is now possible to identify many organic chemicals in drinking water and to measure their concentrations in nanograms per liter. Summarizing reports from different countries, Kraybill (*2*) noted that, of 2221 organic chemicals found in water supplies on a worldwide basis, 765 were present in drinking water. Of these, 20 were recognized carcinogens, 23 were suspected carcinogens, 18 were carcinogenic promoters, and 56 were mutagens. Some of the recognized and suspected carcinogens identified in U.S. drinking water are shown in Table 1. Most of the chemicals reported in these surveys are volatile or low molecular weight organics which can be easily recovered from water (*3*), and these compounds are estimated to account for only 10 to 20% of total organic chemicals present in drinking water. Methods to analyze the nonvolatile organic fraction are still evolving but most of the nonvolatile and high molecular weight compounds remain unidentified.

Table 1. Some recognized and suspected carcinogens identified in U.S. drinking water, 1976.[a]

Chemical	Concentration, μg/L
Aldrin	5.4
Benzene	50.0
Benzo(a)pyrene	0.002
Bis(2-chloroethyl ether)	0.4
Lindane	—
Carbon tetrachloride	3.0
Chlordane	0.1
Chloroform	20–300
1,2-Dibromoethane	—
Dieldrin	8.0
DDT	—
DDE	0.05
1,4-Dioxane	1.0
Endrin	0.004
Heptachlor	—
Trichlorethane	—
Vinyl chloride	10.0

[a] Data: Kraybill (*4*).

Organic chemicals enter water supply systems from a number of major sources. On a quantitative basis, the most important of these sources are industrial discharges, municipal waste water discharges, agricultural runoff, and decomposition of natural organic matter (humus). Other more localized sources include, among oth-

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ers, landfill leachates, accidental spills of chemicals, and leaking underground storage tanks.

Within the past 10 years, investigators have identified several organic by-products of the chlorination disinfection process. Among these compounds are chloroform (a known animal carcinogen), bromoform, bromodichloromethane, and dibromochloromethane. Chloroform invariably occurs in water which has been chlorinated, while it is absent or present at much lower concentrations in unchlorinated groundwater or in raw surface water prior to chlorination (*5*). However, nonchlorinated ground water is by no means free of organic chemicals, as shown in Table 2 (*6*). The significance of this finding is that a number of epidemiologic studies of water quality and cancer risk use populations consuming ground water or nonchlorinated water as a "nonexposed" standard for calculating cancer risk associated with exposure to surface or chlorinated water supplies.

Identification and quantification of the array of organic chemicals in drinking water is a complex and costly procedure and, even now, most of these compounds are rarely measured and none are routinely monitored. Hence, one of the major problems in assessing human health risks associated with chemical contamination of drinking water is the measurement or estimation of human exposure. Can the epidemiologist utilize quantifiable indices of exposure such as levels of chloroform or total organics in water, surrogates of exposure such as chlorinated surface water versus unchlorinated ground water, or direct chemical analysis of specific chemicals? If the latter, how variable are concentrations of these chemicals over time and place, and what is the relevant time interval between exposure and adverse health effect? Can the epidemiologist utilize any biological markers of human exposure to organic chemicals in water?

Epidemiologic Evidence for Adverse Health Effects

Epidemiologic investigations of organic chemicals in water have virtually been limited to considerations of cancer risk. Isolated reports can be found on wells contaminated by leachates from pesticide waste dumps and reputed effects on liver enzyme concentrations in exposed persons (*7*), but systematic epidemiologic studies of the past decade have focused on cancer outcomes. These studies can be divided into two broad categories which are determined by the unit of observation: aggregate risk studies in which geographical areas (counties, cities, states) are the unit of analysis and individual

Table 2. Some synthetic organic chemicals detected in wells used for drinking water.[a]

Chemical	Maximum concentration, μg/L	Location	Evidence for carcinogenicity[b]
Benzene	230	New Jersey	H
α-BHC (α-hexachlorocyclohexane)	6	California	CA
β-BHC	3.8	California	NT
γ-BHC (Lindane)	22	California	CA
Bis(2-ethylhexyl phthalate)	170	New York	NT
Bromoform	20	Delaware	NT
Butyl benzyl phthalate	38	New York	NT
Carbon tetrachloride	400	New Jersey	CA
Chloroform	490	New Jersey	CA
Chloromethane	44	Massachusetts	NT
Cyclohexane	540	New Jersey	NT
DBCP (dibromochloropropane)	137	Arizona	CA
Dibromochloromethane	55	New York	NT
1,1-Dichloroethane	7	Maine	SA
1,2-Dichloroethane	100	New Jersey	CA
1,1-Dichloroethylene	280	New Jersey	NT
1,2-Dichloroethylene	323	Massachusetts	NT
Di-*n*-butyl phthalate	470	New York	NT
1,4-Dioxane	2,100	Massachusetts	CA
EDB (ethylene dibromide, 1,1-dibromoethane)	300	Hawaii	CA
Isopropyl benzene	290	New Jersey	NT
Methylene chloride	47	New York	NT
Parathion	4.6	California	SA
PCE (tetrachloroethylene)	1,500	New Jersey	CA
Toluene	260	New Jersey	NT
1,1,1-Trichloroethane	5,100	New York	NA
1,1,2-Trichloroethane	20	New York	CA
TCE (trichloroethylene)	21,300	Pennsylvania	CA
Trifluorotrichloroethane	135	New York	NT
Vinyl chloride	50	New York	H, CA
Xylene	300	New Jersey	NT

[a] Data of Crump and Guess (*6*). List of chemicals, maximum concentrations, and locations compiled by staff of Council of Environmental Quality (CEQ). [b] H = confirmed human carcinogen; CA = confirmed animal carcinogen; SA = suggestive animal carcinogen; NA = negative evidence of carcinogenicity from animal bioassay; NT = not tested in animal bioassay.

Table 3. Ecological studies of drinking water and cancer.[a]

Investigators	Study location	Data[b]	Exposure variable	Control variables[c]
Harris, 1974 (*9*)	64 Louisiana counties	M	% of county using Mississippi River	U, I, O, D
Buncher, 1975 (*10*)	14 Ohio and 28 Kentucky Counties	M	County served by Ohio River	U, I, O, D
Meinhardt et al., 1975 (*11*)	13 Missouri counties	M	County served by Miss. or Missouri River	D
Page et al., 1976 (*12*)	64 Louisiana counties	M	% of county using Mississippi River	U, I, O
DeRouen et al., 1977 (*13*)	64 Louisiana counties	M	% of county using Mississippi River	U, I, O, location of county
Harris et al., 1977 (*14*)	88 Ohio counties	M	% of county using surface water	U, I, O, D
Kuzma et al., 1977 (*15*)	88 Ohio counties	M	Surface vs. groundwater use	U, I, O, D
Mah et al., 1977 (*16*)	64 Louisiana counties	M,I	Surface vs. ground and chlorination	O, SES, population mobility
Reiches, 1977 (*17*)	187 US counties	M	Surface vs. ground and organic content	U, I, education, industrialization
Salg, 1977 (*18*)	346 counties along the Ohio River	M	% surface water and % chlorination of county	U, I, O, education, mobility, population, % nonwhite, % foreign-born
Cantor et al., 1978 (*19*)	76 US counties	M	THM and chloroform concentrations	U, O, D, education, % foreign, location, population growth
Hogan et al., 1979 (*20*)	US counties	M	Chloroform content of county water	U, I, O, education, % foreign, % nonwhite, location, population
Carlo et al., 1980 (*21*)	Census tracts in Erie County, NY	I	THM concentration of census tract	U, O, SES, mobility, % nonwhite
Tuthill et al., 1980 (*22*)	Massachusetts communities	I	THM concentration and chlorine dose	I, O, SES, mobility, % foreign, % nonwhite, education
Bean et al., 1982 (*23*)	Selected Iowa communities	I	Surface vs. groundwater use	I, O, SES, mobility, education

[a] Data of Callas (*8*).
[b] M = mortality, I = incidence.
[c] U = urbanization; I = income; O = occupation; D = population density.

risk studies. Aggregate risk studies can be performed quite rapidly because public data sources generally provide the required information on cancer mortality, demographic characteristics, and water quality for geographical areas which are the unit of analysis. Fifteen aggregate risk studies were reported for different geographical areas of the U.S. between 1974 and 1982; a recent review of these studies by Callas (*8*) is summarized in Tables 3 and 4. The most common cancer sites statistically associated with various measures of population exposure to organic chemicals in water were bladder, stomach, colon and rectum although other sites showing statistically significant relationships were esophagus, liver, gallbladder, pancreas, kidney, prostate, lung and breast. There was considerable inconsistency among cancer sites associated with water quality in these 15 studies. The problems with drawing causal causal inferences from aggregate risk studies are many and are inherent to the nature of the aggregate unit of analysis: there is great variability of individual exposure within a geographical unit; no individual measurements of exposure are known; potential confounders are seldom known and cannot be linked to individuals; individual exposures cannot be linked to persons who developed cancer. Aggregate risk studies may be useful for justifying more costly epidemiologic investigations of exposed individuals but they leave unresolved the question of causal relationships and provide no basis for quantitative risk assessment. A reasonable conclusion from the reported studies is that there are areas in the U.S. where cancer risk and various (usually surrogate) measures of water quality are associated, and the nature of this association should be investigated by conducting individual risk studies.

One historical cohort study relating water source (groundwater, chlorinated water in small towns, chlorinated water in cities) to cancer incidence for liver, kidney, and bladder was reported from an ongoing investigation of residents of Washington County, MD (*29*). No statistically significant associations with cancer incidence rates were observed, but the power of this study was low, given the relatively small population size (31,000 persons) for calculating incidence rates over a 12-year interval. Thus only 45 cases of liver cancer occurred in the period of followup.

Cantor gave a progress report on a large bladder cancer case-control study conducted by the National Cancer Institute (*30*). From the 10 cancer registries supported by NCI in each of the SEER areas, 3000 incident bladder cancer cases and 6000 controls were interviewed with respect to lifetime residential history, smoking habits, occupation, coffee and artificial sweetener use, and several other factors. From 1000 public water utilities in each of the 10 study areas, the investigators collected historical information on raw water surface versus groundwater or chlorinated vs. nonchlor-

Table 4. Results of ecological studies.[a]

Investigators	Significance[b]	Population[c]	Systems[d]
Harris, 1974 (*9*)	SIG	WM	UT, GI, total
		WF	None
	NS	WM	Liver, lung
		WF	UT, GI, liver, total
Buncher, 1975 (*10*)	NS	WM, WF	Total cancer
Meinhardt et al., 1975 (*11*)	NS	WM, WF, NWM, NWF	UT, GI, liver, lung, total
Page et al., 1976 (*12*)	SIG	WM	UT ($r = 0.66$), GI ($r = 0.62$), total ($r = 0.65$)
		WF	GI ($r = 0.37$)
	NWM	GI ($r = 0.57$), total ($r = 0.61$)	
		NWF	UT ($r = 0.35$), GI ($r = 0.57$), total ($r = 0.57$)
	NS	WM	Prostate
		WF	UT, breast, total
		NWM	UT, prostate
		NWF	Breast
DeRouen et al., 1977 (*13*)	SIG	WM	Total ($r = 0.77$)
		WF	None
		NWM	GI ($r = 0.77$), lung, total ($r = 0.65$)
		NWF	UT ($r = 0.61$), GI ($r = 0.65$), total ($r = 0.73$)
	NS	WM	UT, GI, lung, liver
		WF	UT, GI, liver, total
		NWM	UT, liver
		NWF	Liver
Harris et al., 1977 (*14*)	SIG	WM	Bladder ($r = 0.63$), UT ($r = 0.58$), esophagus ($r = 0.74$), stomach ($r = 0.56$), colon ($r = 0.53$), rectum ($r = 0.53$), GI ($r = 0.61$), lung ($r = 0.77$), total ($r = 0.69$)
		WF	Stomach ($r = 0.37$), liver ($r = 0.40$), breast ($r = 0.62$), total ($r = 0.53$)
	NS	WM	Kidney, pancreas, prostate
		WF	Bladder, UT, esophagus, colon, rectum, GI, kidney, pancreas
Kuzma et al., 1977 (*15*)	SIG	WM	Bladder, stomach, total
		WF	Stomach
	NS	WM	Colon, rectum, pancreas, liver, lung
		WF	Bladder, colon, rectum, pancreas, liver, lung, breast, total
Mah et al., 1977 (*16*)	NS	WM, WF	Kidney, bladder, UT, stomach, esophagus, colon, rectum, GI, liver, lung
Reiches, 1977 (*17*)	NS	WM, WF	Bladder, kidney, UT, stomach, colon, rectum, liver, pancreas, gallbladder, total
Salg, 1977 (*18*)	SIG	WM	Bladder, rectum, reticulosarcoma
		WF	Rectum, breast
		NWM	None
		NWF	Esophagus
	NS	WM	Kidney, esophagus, stomach, colon, liver, pancreas, lung, prostate, total, other sites
		WF	Bladder, kidney, esophagus, stomach, colon, liver, pancreas, lung, total, other sites
		NWM	Bladder, kidney, esophagus, stomach, colon, rectum, liver, pancreas, lung, prostate, total, other sites
		NWF	Bladder, kidney, stomach, colon, rectum, liver, pancreas, lung, breast, total, other sites
Cantor et al., 1978 (*19*)	SIG	WM	Bladder ($r = 0.38$), kidney ($r = 0.42$)
		WF	Bladder ($r = 0.45$), lung ($r = 0.22$)
	NS	WM	Esophagus, stomach, colon, rectum, liver, lung, total, other sites
		WF	Kidney, esophagus, stomach, colon, rectum, liver, total, other sites
Hogan et al., 1979 (*20*)	SIG	WM	Colon, rectum
		WF	Bladder, colon, rectum
	NS	WM	Bladder, kidney, esophagus, stomach, liver, pancreas, total, other sites
		WF	Kidney, esophagus, stomach, liver, pancreas, total, other sites
Carlo et al., 1980 (*21*)	SIG	WM	Pancreas ($r = 0.16$)
		WF, NWM, NWF	None
	NS	WM	Bladder, esophagus, stomach, colon, rectum
		WF, NWM, NWF	Bladder, esophagus, stomach, colon, rectum, pancreas
Tuthill et al., 1980 (*22*)	SIG	M	Rectum ($r = 0.50$), stomach ($r = 0.60$)
		F	Stomach ($r = 0.50$)
	NS	M	Bladder, kidney, esophagus, colon, liver, pancreas, lung, other sites

(continued)

Table 4 (continued)

Investigators	Significance[b]	Population[c]	Systems[d]
		F	Bladder, kidney, esophagus, colon, rectum, liver, pancreas, lung, breast, other sites
Bean et al., 1982 (*23*)	SIG	WM	Rectum, lung
		WF	Rectum, lung
	NS	WM	Bladder, colon, stomach, prostate
		WF	Bladder, colon, stomach, breast

[a] Data of Callas (*8*).
[b] Significance: SIG = significant association between drinking water and cancer; NS = no significant association.
[b] Population: WM = white male; WF = white female; NWM = nonwhite male; NWF = nonwhite female.
[d] Systems: UT = total urinary tract cancer; GI = total gastrointestinal cancer; r = correlation coefficient (reported if given in study).

inated water at place of usual residence. Significant association with water quality were found for: mortality from cancer of the bladder in two of five studies, cancer of the colon in three of five, and cancer of the rectum in four of five case-control reports.

Five individual risk studies, all of them case-control in design, have been reported in the literature (*9*) and are reviewed in Tables 5 and 6. Each of the five studies obtained cases and controls from state death registries and utilized data on exposure and potential confounders from information on the death certificate and secondary public sources. Since relatives of decedents were not interviewed, the investigators had no knowledge of smoking or dietary habits, residential mobility of individuals, personal consumption habits, or other factors which may have affected cancer risk. Crude surrogates of exposure to organics in water were utilized, such as sources, treatment practices, and geographical areas served. Approximately 50% of persons in one area (Iowa) served in 1975 by chlorinated surface or groundwater sources had a different water source in 1955, demonstrating that recent water sources could lead to significant misclassification of exposure status in this area if exposures of 20 years ago were the most relevant to cancer risk. The overall results of this major study have not been reported to date.

The State of Knowledge and Future Directions

At this point in time, the epidemiologic evidence on the issues of organic chemicals in water and cancer risk is no more than embryonic. Some associations found for aggregate units of analysis were also observed in a few case-control studies. Exposure to a crude measure of organic water quality appears to be associated with a small but signficantly increased risk of cancer of the colon, rectum and/or bladder. The studies to date are neither consistent in finding a relationship with one or the same set of cancer sites, nor did they convincingly control for potential confounding factors. Hence one cannot conclude that a causal relationship is by any means established. However, the generally positive nature of

Table 5. Case-control studies of drinking water and cancer.[a]

Investigators	Cases[b]	Controls	Exposure	Control variables[c]
Alavanja et al., 1979 (*24*)	3446 UT and GI cancer deaths in 7 New York counties, 1968–1970	Noncancer deaths from same years matched on age, race, sex, foreign vs. U.S. born, county of residence	Surface vs. ground and chlorination vs. nonchlorination of water source at residence	U, O
Struba, 1979 (*25*)	Bladder, colon, rectum cancer deaths in North Carolina, 1975–1978 (700–1500 cases/cancer site)	Noncancer deaths matched on age, race, sex, residence in region of North Carolina	Surface vs. ground and chlorination vs. nonchlorination of water source at residence	U, O, SES, place of birth
Brenniman et al., 1980 (*26*)	3208 UT and GI cancer deaths in Illinois, 1973–1976	43,666 nonmatched noncancer deaths in Illinois, 1973–1976	Chlorinated vs. nonchlorinated groundwater residence	Age, race, sex, urban vs. rural residence, population of community
Young et al., 1981 (*27*)	8029 female cancer deaths in Wisconsin, 1972–1977	Noncancer deaths matched on age, race, sex, year of death, county of residence	Chlorination of water, surface vs. ground	U, O, marital status
Gottlieb et al., 1982 (*28*)	11,349 deaths from 11 cancer sites in 20 counties of southern Louisiana, 1960–1975	Noncancer deaths matched on age, race, sex, year of death, county group	Surface vs. ground and chlorination vs. nonchlorination of water source for residence	U, O, industrialization of county

[a] Data of Callas (*8*).
[b] UT = total urinary tract cancer, GI = total gastrointestinal cancer.
[c] U = urbanization, O = occupation.

Table 6. Results of case-control studies.[a]

Investigators	Significance[b]	Population[c]	Systems[d]
Alavanja et al., 1979 (*24*)	SIG	M	Bladder (OR = 2.02), liver and kidney (OR = 2.76), esophagus (OR = 2.39), stomach (OR = 1.67), colon (OR = 1.99), rectum (OR = 2.33), pancreas (OR = 2.23), total cancer (OR = 1.44)
		F	Stomach (OR = 2.23), total cancer (OR = 1.44)
	NS	M	Lung
		F	Bladder, liver and kidney, esophagus, colon, rectum, pancreas, lung
Struba, 1979 (*25*)	SIG		Bladder (OR = 1.54), colon (OR = 1.30), rectum (OR = 1.54)
	NS		None
Brenniman et al., 1980 (*26*)	SIG	M	None
		F	Rectum (OR = 1.35), total GI (OR = 1.15)
	NS	M	Bladder, other urinary organs, esophagus, stomach, colon, rectum, total GI, liver, gallbladder, pancreas
		F	Bladder, other urinary organs, esophagus, stomach, colon, liver, gallbladder, pancreas
Young et al., 1981 (*27*)	SIg		Colon (OR = 1.35)
	NS		Bladder, kidney, esophagus, stomach, rectum, liver, pancreas, lung, brain, breast
Gottlieb et al., 1982 (*28*)	SIG		Rectum (OR = 1.68), breast (OR = 1.58)
	NS		Bladder, kidney, esophagus, stomach, colon, liver, pancreas, lymphoma, leukemia, melanoma, multiple myeloma, brain, prostate

[a] Data of Callas (*8*).
[b] Significance: SIG = significant association between exposure and cancer; NS = no significant association.
[c] Population: M = male; F = female.
[d] Systems: UT = urinary tract; GI = gastrointestinal tract; OR = odds ratio.

the associations continues to justify further pursuit of the issue.

The question to be addressed is, what further evidence should we seek, or where should new epidemiologic investigations go to shed light on the issue. Further aggregate risk studies are likely to be nonproductive. Clearly, we need some large case-control interview studies and cohort studies that might be fortuitously superimposed on other research objectives. Given the rare frequency of bladder and rectal cancer, a cohort study will have to involve many person-years of followup, since the incidence of these tumors is on the order of 10 to 20 per 100,000. However, the complexity of assessing exposure to organics in water may entirely preclude the conduct of any cohort study of cancer risk.

Assessment of relevant individual exposure to organics in water remains the thorny barrier to progress. The usual surrogates of exposure based on surface versus groundwater or chlorinated water versus unchlorinated water sources are unsatisfactory, given accumulating evidence for an array of organics in ground and unchlorinated drinking water. The resulting misclassification of exposure status, if nondifferential among cases and controls, will bias risk estimates towards the null hypothesis, and this bias may be severe. If surrogate measures are to be used (and alternative measures may not be justifiable until much more is known about the chemistry of organic contamination of water supplies), the epidemiologist will have to hammer out a better exposure index by consulting with knowledgeable water chemists and engineers. Questions to be addressed include:

- How can surface water be graded with respect to potential contamination with organic chemicals?
- How can knowledge of upstream dischargers (for which data sources are available) be incorporated into the above question?
- Is the method of sedimentation, filtration, and other water treatment relevant to the formation of potentially harmful by-products of chlorine disinfection?
- Is there a predicable relationship between amount of chlorine added and chlorine residual (for which historical data exist) and concentration of chlorinated by-products?
- Is well depth relevant to potential exposure to organic chemicals?
- Does proximity of wells or of underground aquifers to agricultural runoff affect the water quality of these sources?
- Is there considerable temporal variation in organic constituents and concentrations for the same surface and groundwater source over the span of one year and of several years?

If some or all these questions were even partially answered, it would be possible to design interview studies which, combined with historical data available at each water utility, could lead to much more refined sur-

rogate measures of exposure to organic chemicals in water. These surrogates might then reasonably be validated, at least for currently obtainable water samples, by comparison with direct chemical analyses.

Exposure estimates based on direct identification and quantification of organics in water seems an unfeasible approach to epidemiologic studies, given the large array of chemicals, the uncertainty of cancer risk related to nearly all of the chemicals, the multistage process of carcinogenesis, and the possibility of interaction between chemicals. Complexity and cost of chemical analyses would also seem to argue against direct measurements for individual risk studies.

A potentially fertile approach for population studies, especially for the limited sample sizes required in case-control studies, is that of biological markers of exposure. More than 100 volatile organics can be readily isolated from human blood and other body fluids (*3*). Wallace et al. (*31*) claim that breath samples, analyzed for volatile organics, reflect personal exposures to target chemicals in water and in air. Profiles of chemical adducts to DNA can also be obtained from individual blood samples. Considerable work needs to be done to related intake of known organics to these biological markers before the markers themselves could be judged feasible for epidemiologic investigations.

It appears that the immediate need is for a collaborative research effort, between epidemiologists, analytical chemists, and water quality engineers, to characterize human exposure to organics in water. This collaboration needs to be directed by the epidemiologists's research objectives, specifically relating cancer risk at least to an ordinal ranking of cancer relevant exposures. If a biological marker could best characterize this exposure, all the better since this would provide a personal integrated exposure estimate. Further epidemiologic studies of cancer risk and water quality, in the absence of progress of exposure estimation, seems unlikely to advance our knowledge of the nature of this relationship.

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